## CORRECTION

An abbreviation was incorrectly defined by the copyeditor in an article in the April issue of the *Journal* (Volume 106, No. 4, page 631).

The abstract of the article by Hatamochi *et al*, "Collagenase gene expression in cutis laxa fibroblasts is upregulated by transcriptional activation of the promoter gene through a 12-O-tetradecanoyl-phorbol-13-acetate (TPA)-responsive element," is reprinted below.

**Our previous work demonstrated that collagenase mRNA levels are increased in fibroblasts derived from patients with cutis laxa (CL). To pursue the mechanism of the upregulation of collagenase expression, we investigated transcriptional levels of the collagenase gene in CL fibroblasts. Fibroblasts cultured from the skin of three congenital CL patients were studied. Northern blot hybridization revealed 2.8– to 7.3–fold increases in collagenase mRNA levels in CL fibroblasts compared with normal cells. Nuclear run-off experiments demonstrated that the transcription rate of the collagenase gene in nuclei isolated from the same cells was 5.1– to 10.2–fold higher in the CL fibroblasts than in the controls. Transient transfection of a normal collagenase promoter–CAT construct into the cells further showed significantly enhanced transcriptional activity in CL but not in normal fibroblasts. Experiments of transient transfection of deleted or small substituted collagenase promoter–CAT constructs indicated that collagenase transcription in CL fibroblasts was activated through the TPA-responsive element site of the collagenase promoter gene. Although the levels of *Jun* and *Fos* gene expression did not differ from those observed in normal fibroblasts, AP-1-binding activity, as measured by the ability to bind to an oligonucleotide containing a TPA-responsive element, was significantly elevated in CL fibroblasts as compared with normal fibroblasts. These data suggest that collagenase expression is upregulated at the transcriptional level by endogenous activation of DNA binding of AP-1 in CL fibroblasts. *Key words: matrix metalloproteinase-1. J Invest Dermatol 106:631–636, 1996***

## CORRECTION

There was a typographical error in a table in an article in the April issue of the *Journal* (Volume 106, No. 4, page 736).

Table I from the article by Marinkovich *et al*, "LAD-1, the linear IgA bullous dermatosis autoantigen, is a novel 120-kDa anchoring filament protein synthesized by epidermal cells," is reprinted correctly below.

**Table I. The 123 Antigen Shows a Unique Tissue Distribution***

| Tissue | 123 Antigen | Laminin-5 | Collagen VII | Laminin-1 |
|---|---|---|---|---|
| Skin | ++ | ++ | ++ | ++ |
| Cornea | ++ | ++ | ++ | ++ |
| Lung | − | ++ | ± | ++ |
| Esophagus | ++ | ++ | ++ | ++ |
| Small intestine | + | ± | ± | ++ |
| Ureter | ++ | ++ | ++ | ++ |
| Bladder | ++ | ++ | ++ | ++ |
| Thymus | ++ | ++ | ++ | ++ |
| Peripheral Nerve | − | − | − | ++ |
| Skeletal muscle | − | − | − | ++ |
| Blood vessels | − | − | − | ++ |

* A 24-inch crown to rump length fetal bovine calf was used for all tissues except amnion which was human and obtained from a normal term delivery. Indirect immunofluorescent microscopy was performed on frozen sections of all tissues using antibodies against the indicated proteins as described in *Materials and Methods*. ++, strong staining; +, weak staining; ±, negligible staining; −, absent staining.